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Sexual selection can constrain sympatric speciation

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Recent theory has suggested that sympatric speciation can occur quite easily when individuals that are ecologically similar mate assortatively. Although many of these models have assumed that individuals have equal mating success, in nature rare phenotypes may often suffer decreased mating success. Consequently, assortative mating may often generate stabilizing sexual selection. We show that this effect can substantially impede sympatric speciation. Our results emphasize the need for data on the strength of the stabilizing component of selection generated by mating in natural populations.

Keywords: sympatric speciation; sexual selection; assortative mating

1. INTRODUCTION

Of the many mechanisms that have been proposed for sympatric speciation, one of the simplest was sketched by Darwin (1859). He argued that two new species can emerge if intraspecific competition erodes less fit intermediate phenotypes. With the integration of Mendelism and Darwinism, it was realized that sympatric speciation also requires some form of non-random mating to offset the genetic homogenization caused by sexual reproduction.

Darwin's hypothesis has been rejuvenated by genetic models that suggest that the joint action of ecological competition and assortative mating can readily cause sympatric speciation (Doebeli 1996; Dieckmann & Doebeli 1999; Kondrashov & Kondrashov 1999; Doebeli & Dieckmann 2000, 2003). An attractive feature of these models is that they include plausible forms of competition that can produce frequency-dependent disruptive selection when the population is at equilibrium.

A key assumption of several of these models is that individuals have equal reproductive success, a situation we refer to as 'non-selective mating'. Mating success in most species is highly variable, however, generating ample opportunity for sexual selection (Bateman 1998; Beeching & Hopp 1999; Harari *et al.* 1999). Assortative mating may typically contribute a stabilizing component to lifetime selection because rare phenotypes are often less likely to find mates than are common phenotypes. Here, we show that this effect can greatly decrease the potential for sympatric speciation under Darwin's hypothesis. Stabilizing sexual selection generated by assortative mating can work against sympatric speciation by causing fixation at individual loci and by reducing the associations between alleles at different loci (linkage disequilibria) that are the genetic basis for sympatric speciation. Many authors have noted that mating may penalize rare phenotypes and make sympatric speciation less likely (Karlin & Scudo 1969; Kondrashov & Mina 1986; Noest 1997; Kondrashov & Shpak 1998; Van Doorn *et al.* 1998; Dieckmann & Doebeli 1999; Kondrashov & Kondrashov 1999; Drossel & McKane 2000; Takimoto *et al.* 2000). The impact of this effect on polymorphism and the implications for sympatric speciation under Darwin's hypothesis, however, seem not to be fully appreciated.

Here, we present models in which sympatric speciation is mediated by a single trait that experiences disruptive natural selection and that is the basis for assortative mating. We consider two forms of assortative mating, which represent plausible scenarios for different groups of organisms, and study them using both analytic approximations and numerical simulations.

2. SELECTION AND MATING

In general form, our assumptions follow those of Doebeli (1996). A single trait is subject to stabilizing abiotic selection and to disruptive selection generated by intraspecific competition. This same trait is the basis for assortative mating. We assume either that the population is hermaphroditic or, if it is dioecious, that the trait is expressed equally in males and females. The density of individuals with phenotype x is written $f(x)$, and the variance of the population is V. The life cycle begins with competition, followed by abiotic selection and finally mating.

(a) *Competition*

Sympatric speciation requires some form of diversifying selection. In Darwin's hypothesis, disruptive selection is generated by intraspecific competition. We assume that the intensity of competition between two individuals with phenotypes x and y declines as a Gaussian function of the difference between them (Slatkin 1980; Doebeli 1996). The competitive fitness of an individual with phenotype x, $W_c(x)$, is determined by the average amount of competition it experiences in the population:

$$W_c(x) = 1 - c_1 \int f(y) \mathrm{Exp}\{-c_2(x-y)^2/2\}\,\mathrm{d}y. \qquad (2.1)$$

The term in braces is the competitive effect that individuals with phenotypes x and y have on each other. The parameter c_1 is a frequency-independent component that measures how much two identical individuals decrease each other's fitnesses. The parameter c_2 measures the

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specificity of the competition (i.e. a large value means that only phenotypically similar individuals compete).

(b) *Abiotic selection*

Traits that mediate assortative mating are typically subject to stabilizing selection from the abiotic environment. (This type of selection can also come from the biotic environment, of course, but we use 'abiotic' as a simple way to distinguish it from selection generated by intraspecific competition.) We assume that $W_a(x)$, the fitness from abiotic selection of an individual with phenotype x, is a Gaussian fitness function with an optimum at 0 and a variance of $1/a$; individuals whose phenotypes are within $1/\sqrt{a}$ of the optimum have high fitness. Thus the intensity of abiotic stabilizing selection is measured by a.

(c) *Assortative mating*

We will investigate two simple scenarios for assortment, which we call the 'plant model' and the 'animal model'. In both, the relative attraction, $A(x,y)$, between individuals with phenotypes x and y declines as a Gaussian function of the difference between them:

$$A(x,y) = \mathrm{Exp}\{-m(x-y)^2/2\}. \tag{2.2}$$

Here, m measures the strength of assortative mating. Roughly speaking, individuals whose phenotypes differ by less than $2/\sqrt{m}$ have a high probability of mating if they meet.

The difference between our plant and animal models involves reproductive assurance. In the plant model, the probability of two individuals mating is simply proportional to A. The frequency of matings between all x females and y males in the population is the product of the frequencies of those phenotypes, weighted by A and normalized so that the frequencies total 1:

$$M(x,y) = \frac{f^*(x)\,f^*(y)A(x,y)}{\iint f^*(x)\,f^*(y)A(x,y)\mathrm{d}x\mathrm{d}y}, \tag{2.3}$$

where $f^*(x)$ is the density of phenotype x after competition and abiotic selection. Mating success through both male and female function depends here on the frequency of similar individuals in the population. This is a plausible model for assortment based, for example, on flowering time when pollen is limiting: both male (pollen) and female (ovule) reproductive successes depend on encountering other gametes that are available to fuse at the same time.

Under the animal model, the probability that two individuals mate follows a similar rule except that females are assured of mating. The frequency of matings is now normalized so that the frequency of mated pairs involving an x female is equal to $f(x)$, the frequency of such females in the general population:

$$M(x,y) = \frac{f^*(x)\,f^*(y)A(x,y)}{\int f^*(y)A(x,y)\mathrm{d}y}. \tag{2.4}$$

This model is applicable to polygynous animal populations where females search until they find an acceptable mate, and to plant populations when pollen is not limiting.

A critical feature of both the plant and animal models for mating is that they result in positive frequency dependence. Rare phenotypes are less likely to find mates than are common ones. We will see that this kind of sexual selection can have important consequences for sympatric speciation.

(d) *Lifetime fitness*

The expected lifetime fitness of an individual with phenotype x, $W_T(x)$, is

$$W_T(x) = W_c(x)\,W_a(x)\left[\frac{1}{2}\int\frac{M(x,z)}{f^*(x)}\mathrm{d}z + \frac{1}{2}\int\frac{M(z,x)}{f^*(x)}\mathrm{d}z\right]. \tag{2.5}$$

The quantity in square brackets, which represents reproductive success, is the expected fitness achieved through female function (the first term) and that achieved through male function (the second term), averaging over the probability of mating with different phenotypes. Under the animal model, the first integral equals 1, reflecting the assumption that females have equal mating success. Under the plant model, the two terms inside the brackets are identical, as reproductive successes through male and female functions are equal.

3. DISRUPTIVE SELECTION: A PRECONDITION FOR SPECIATION

Sympatric speciation requires that disequilibria between the alleles that contribute to the trait build up to the point where a unimodally distributed population splits into two. While this process can be driven by non-random mating alone, the conditions are stringent, and generally disruptive natural selection is also needed (Kondrashov & Shpak 1998).

Disruptive selection is important to some recent models of sexual selection for a second reason. Several of them imagine that the process begins with allele frequencies of 1/2 at all loci for the traits that mediate mating (Dieckmann & Doebeli 1999; Higashi *et al.* 1999). This situation is highly favourable to speciation, but requires quite special conditions. Wright (1935) showed that, under random mating, if lifetime fitness produces weak stabilizing selection on a trait that is determined by loci with additive effects, then selection can at best maintain polymorphism at only one of the loci. That is, stabilizing selection will favour fixation at all or all but one of the loci. While other forces such as mutation can maintain variation even when selection favours fixation, allele frequencies will typically be far from 1/2, making sympatric speciation under Darwin's hypothesis much more difficult.

Wright's result generalizes directly to non-random mating. When selection is weak, the force of selection, a_i, acting directly on a locus i that contributes to a trait under stabilizing selection near an optimum at $x = 0$ is

$$a_i = C\frac{b_i}{\bar{W}}\left[\bar{x} + \frac{1}{2}b_i(q_i - p_i)\right], \tag{3.1}$$

where C is the curvature (second derivative) of the lifetime fitness function, b_i is the effect of alleles at locus i on the trait, $\bar{W}$ is the population's mean fitness, $\bar{x}$ is the trait mean, and p_i and q_i are the frequencies of the alleles

(Barton 1986). This result holds regardless of the mating system (Kirkpatrick *et al.* 2002). The first term inside the square brackets is the force of selection pushing the population mean towards the optimum. The second term is the effect of selection on the variance. This force favours fixation of the common allele when selection is stabilizing overall ($C < 0$), but maintains variation when selection is disruptive.

We can therefore learn about conditions favourable to sympatric speciation by asking when lifetime fitness results in disruptive selection. Consider a population whose mean lies at the abiotic optimum of 0, and assume that selection and assortment are weak, specifically that c_1c_2V, aV and $mV \ll 1$. (Under these conditions, linkage and Hardy–Weinberg disequilibria will be weak, and sympatric speciation is not possible. Simulation results shown in § 4, however, suggest that the approximation is quite accurate even when these conditions are violated.) Approximating the fitness functions by quadratic equations and calculating the integrals of equations (2.1) and (2.5) then shows that under the plant model of assortative mating

$$W_{\mathrm{T}}(x) \approx 1 - c_1 + \frac{1}{2}[c_1c_2 - (1 - c_1)(a + m)]x^2. \qquad (3.2)$$

For the animal model, m is replaced by $m/2$. The terms inside the square brackets represent the contributions of competition, abiotic selection and sexual selection to the shape of the fitness function. Competition generates disruptive selection, reflected by the fact that increasing values of c_1 and c_2 contribute positively to the coefficient of x^2, while abiotic and sexual selection are both stabilizing.

Equation (3.2) shows that, if competition is weaker than the combined strength of abiotic selection and assortment, then lifetime fitness causes stabilizing selection. In that event, selection will tend to fix all or all but one of the loci. Conversely, the condition for disruptive selection is that

$$a + km < \frac{c_1c_2}{1 - c_1}, \qquad (3.3)$$

where $k = 1$ for the plant model and $k = 1/2$ for the animal model.

The simple but important conclusion from equations (3.2) and (3.3) is that increasing the intensity of assortment (larger values of m) increases the intensity of stabilizing selection, which stymies sympatric speciation. This is perhaps counterintuitive at first look, as assortment is also the critical factor that makes sympatric speciation possible. Stabilizing selection produced by sexual selection in the animal model is half as intense as in the plant model for the simple reason that half of the population (the females) are assured equal reproductive success.

4. CONDITIONS FOR SYMPATRIC SPECIATION

Condition (3.3) is not sufficient for sympatric speciation under Darwin's hypothesis. Speciation requires that associations between alleles (linkage and Hardy–Weinberg disequilibria) build up to a level at which the population splits into two modes. Assortment and disruptive natural selection resulting from competition increase these associations, but stabilizing selection caused by abiotic selection and mating decrease them. How will these conflicting forces play out?

We can use the quasi-linkage equilibrium approximation developed by Barton & Turelli (1991) and Kirkpatrick *et al.* (2002) to get analytic results when selection and assortment are weak. Assume that two alleles with frequencies p_i and q_i segregate at diploid locus i. Their effects are additive (no dominance or epistasis), and the difference in their effects on the phenotype is denoted by b_i. The approximation requires that linkage is not very tight (specifically that (r_{ij}/b_ib_j) is very much larger than a, $c_1c_2/(1 - c_1)$ and m, where r_{ij} is the recombination rate between loci i and j). Electronic Appendix A finds that, at equilibrium, the change in the trait variance caused by selection and non-random mating is

$$\delta V \approx 2m\sum_i b_i^4 p_i^2 q_i^2 + 2m\sum_{i \neq j}\sum b_i^2 b_j^2 p_i q_i p_j q_j + 2\sum_{i \neq j}\sum b_i^2 b_j^2 p_i q_i p_j q_j \left[\frac{c_1c_2 - a - km}{r_{ij}} + m\right], \qquad (4.1)$$

where again $k = 1$ for the plant model and $k = 1/2$ for the animal model. The three terms summed on the right are the contributions from, respectively, Hardy–Weinberg disequilibria within loci, associations between alleles at different loci that were inherited from different parents (between-locus Hardy–Weinberg disequilibria) and gametic disequilibria.

The main conclusion from equation (4.1) is that, with free recombination ($r_{ij} = 1/2$), increasing assortment (larger m) leads to greater variance and therefore pushes the population towards sympatric speciation. A modest decrease in the recombination between a pair of loci ($r_{ij} < 1/3$ for the plant model, $r_{ij} < 1/6$ for the animal model when $p_i = p_j = 1/2$ and $b_i = b_j$) suffices to reverse that trend, however, so that stronger assortment actually decreases the overall variance. Thus tighter linkage inhibits the potential for sympatric speciation.

If it occurs, the full process of sympatric speciation requires strong selection and assortment. Because our analytic approximations do not apply to those situations, we developed a simple multilocus model, which we studied by simulation. There are n unlinked diploid loci that have equal and additive effects on the phenotype. For simplicity, we assume that the trait has perfect heritability. To maximize the opportunity for sympatric speciation, we assume that an individual that is heterozygous at all loci has a phenotype that lies at the abiotic optimum ($x = 0$), so that the mean of a population with the maximal amount of genetic variation (allele frequencies equal to 1/2 at all loci) also lies at the abiotic optimum. It is convenient to define the scale of measurement so that the range of phenotypic values is 1.

The simulations iterated the frequencies of the 2^{2n} genotypes deterministically. This procedure departs from several previous studies based on the 'hypergeometric' model that constrains allele frequencies to be equal at all loci (e.g. Doebeli 1996; Kondrashov & Shpak 1998; Kondrashov & Kondrashov 1999; Takimoto *et al.* 2000). That assumption can artificially stabilize the equilibrium (Barton & Shpak 2000). The cost of our approach is that the speed of the simulations rapidly declines with increasing n, and so we were able to study outcomes with only small numbers of loci. By neglecting the effects of drift,

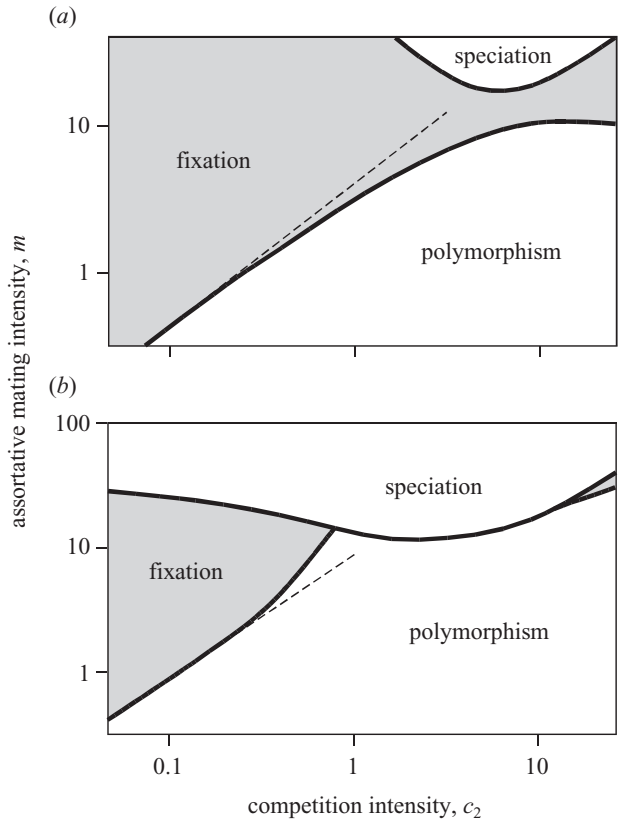

Figure 1. Combinations of the intensities of assortative mating (m) and competition (c_2) that lead to fixation, polymorphism and sympatric speciation under (*a*) the plant and (*b*) the animal models for selective mating. Dashed lines are from the analytic approximation (equation (3.3)) above which fixation is predicted. Solid curves were determined from deterministic multilocus simulations. Parameter values are $n = 4$, $a = 0$ and $c_1 = 0.8$. Note the log–log scale.

which make fixation more likely, we bias the simulations in favour of predicting sympatric speciation.

Simulations began with a unimodal population in linkage and Hardy–Weinberg equilibrium, with allele frequencies at each locus set to 1/2 plus a small random deviate. This initial condition is implausible when selection is initially stabilizing, as we discussed in § 3. We chose this initial condition to maximize the potential for sympatric speciation, so the results are conservative in the sense that we expect sympatric speciation to be even less likely than the simulations suggest.

First, we asked how well the analytic approximation (equation (3.3)) for the maintenance of polymorphism performs. The equilibrium at $p_i = q_i = 1/2$ was deemed to be stable if all allele frequencies evolved towards 1/2 after 100 generations. Results show that equation (3.3) is a good approximation when selection is weak and often does well when it is not. Figure 1 shows the case of $n = 4$ loci (producing a genetic variance of $V = 1/32$ in a randomly mating population with no selection), with no abiotic stabilizing selection ($a = 0$) and a strong frequency-independent component of competition ($c_1 = 0.8$). We see that equation (3.3) quite accurately predicts when fixation occurs for values of c_2 of less than 1 for the plant model of assortment. With the animal model, the approximation does well for c_2 less than 0.5 so long as m is not very large (more than 10).

A basic conclusion is that fixation does indeed preclude sympatric speciation over a substantial portion of the parameter space when there is selective mating (figure 1). As anticipated from the analytic approximations, the plant model is particularly restrictive: fixation can occur even when there is intense disruptive selection resulting from competition.

Second, we used the simulations to determine when sympatric speciation will occur. To compare outcomes under selective and non-selective mating, we developed a deterministic simulation model using the assortment rule given in equation (2.2) and the algorithm for monogamous pairing described in the appendix of Kirkpatrick *et al.* (1990).

We say that speciation occurs when a bimodal equilibrium is reached, even though complete reproductive isolation is not achieved and a very low frequency of intermediate phenotypes persists at equilibrium. This is therefore a conservative criterion in the sense that it favours a finding of sympatric speciation. Examples of cases that do and do not lead to speciation are shown in figure 2.

To make the results easier to interpret, it is helpful to recast the intensity of assortment, m, in terms of ρ, the correlation between mated pairs. This in turn depends on the distribution of genotypes in the population (in particular, the correlation grows as the variance expands). A calculation shows, at the beginning of the process, when the population is at Hardy–Weinberg and linkage equilibrium, that the correlations for animal and plant models are

$$\rho_{\text{animal}} = \frac{m}{\sqrt{m^2 + 8nm + 64n^2}}, \quad \rho_{\text{plant}} = \frac{m}{m + 8n}. \tag{4.2}$$

Figure 1 and table 1 show the results. When the intensity of disruptive selection resulting from competition is weak ($c_2 < 0.5$), sympatric speciation did not occur in the plant model for any intensity of assortment that we simulated ($m < 40$). Speciation becomes possible with strong disruptive selection from competition ($c_2 = 5$). In this case, the survival of individuals in the centre of the distribution is about half that of individuals with extreme genotypes at the start of the process. With $n = 4$ loci, speciation requires that the correlation among mates must be at least $\rho = 0.40$. Recall that we have assumed that the trait is perfectly heritable. If we include environmental variation, the phenotypic correlations between mates needed for speciation are found by dividing the entries in table 1 by h^2, the trait's heritability. Thus with a heritability of 0.5, the best situation for the plant model shown in table 1 requires a phenotypic correlation between mates of 0.66; with h^2 less than 1/3, sympatric speciation is not possible.

Figure 1 shows that speciation can occur when the condition for disruptive selection (equation (3.3)) is violated if competition and assortment are very strong. In these situations, the fitness function is initially stabilizing but changes to disruptive. That is because mating produces positive frequency dependence. With strong assortment, large linkage disequilibria rapidly build up, increasing the population variance. A critical threshold is reached at which intermediate phenotypes now have lower fitness than more extreme ones. Selection is then disruptive, which, when combined with the disequilibria favoured by

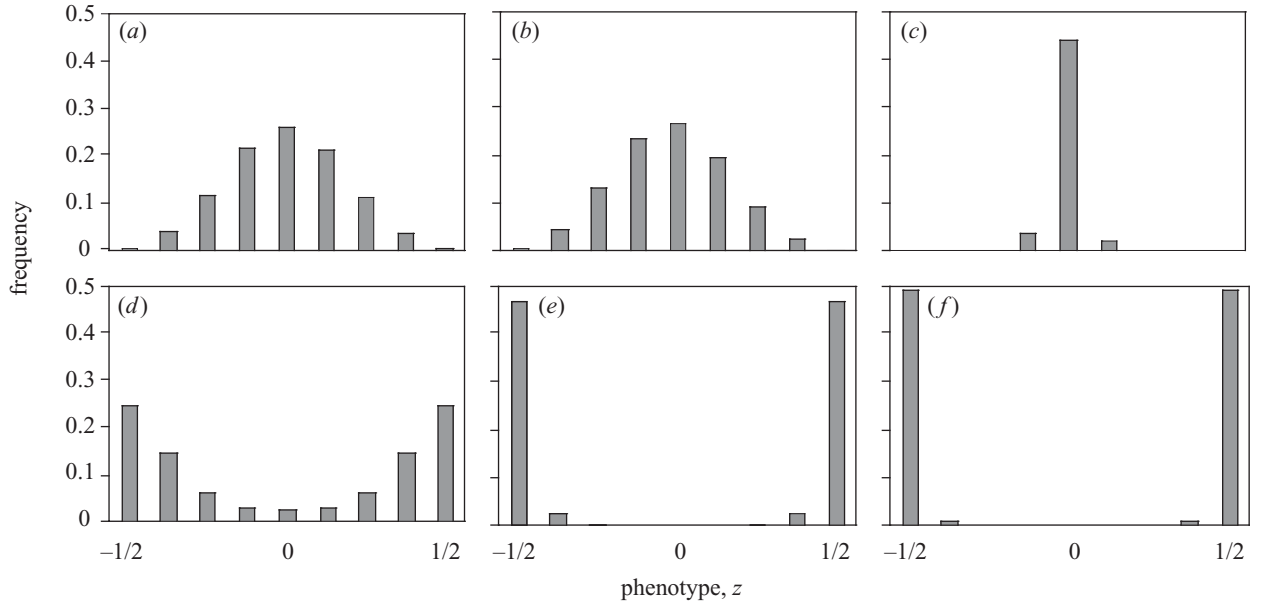

Figure 2. Comparison of the evolutionary dynamics under selective (*a–c*) and nonselective (*d–f*) mating. The non-selective mating population speciates rapidly, whereas the selective-mating population becomes fixed at all loci and fails to speciate. (*a*,*d*) Generation 40; (*b*,*e*) generation 120; and (*c*,*f*) generation 200. Parameter values are $n = 4$, $a = 0$, $c_1 = 0.8$, $c_2 = 0.5$, $m = 15$, with the plant model of selective mating.

Table 1. Critical values for the strength of assortment, m, and the initial correlation between mates, ρ, (in parentheses) required for sympatric speciation under non-selective mating and under the animal and plant models of selective mating.
(Dashes indicate that sympatric speciation does not occur. Parameter values are $a = 0$ and $c_1 = 0.8$. Entries for m have a precision of ±0.5.)

	$n = 2$			$n = 3$			$n = 4$		
c_2	non-selective	animal	plant	non-selective	animal	plant	non-selective	animal	plant
0.005	18.5 (0.62)	15.5 (0.57)	—	24.5 (0.58)	24.0 (0.58)	—	33.0 (0.59)	33.5 (0.59)	—
0.05	16.5 (0.59)	12.5 (0.51)	—	20.0 (0.52)	19.5 (0.52)	—	24.0 (0.49)	27.5 (0.53)	—
0.5	12.5 (0.51)	10.5 (0.45)	—	13.5 (0.41)	12.5 (0.39)	—	14.0 (0.34)	17.0 (0.39)	—
5.0	16.0 (0.58)	11.5 (0.48)	10.0 (0.38)	16.5 (0.47)	12.5 (0.39)	12.0 (0.33)	16.5 (0.39)	13.0 (0.32)	17.5 (0.35)

assortment, causes the population to fission into two modes.

Speciation in these situations thus involves a race between the build-up of disequilibria, which can lead to disruptive selection and sympatric speciation, and the fixation of alleles by stabilizing selection, which can prevent it. The race is strongly biased towards the sympatric-speciation outcome when all loci begin with intermediate allele frequencies, as in our simulations. With less symmetric (and more biologically plausible) initial allele frequencies, fixation will occur over a much wider range of initial conditions. Thus whether or not speciation occurs is strongly dependent on the initial conditions (Kirkpatrick & Ravigné 2002). We expect that the potential for sympatric speciation will be greater when the number of loci is large as the strength of selection on individual loci favouring fixation decreases.

The simulations show three more patterns that seem at first to be counterintuitive. All involve such large parameter values that they may not be biologically relevant. First, the critical value of non-random mating needed for speciation is not a monotonic function of the intensity of competition, c_2 (figure 1). That is because when c_2 is large increasing its value further actually decreases the effective strength of disruptive selection because individual genotypes compete equally weakly with all other genotypes. Second, non-selective mating sometimes requires stronger assortment (a larger value of m) for sympatric speciation than does selective mating (table 1). This outcome happens when competition is so extreme that the population is bimodal after competition but before mating. Then selective mating actually generates additional disruptive selection, which facilitates sympatric speciation. Third, some simulations show that the phenotypic distribution can break into more than two modes (results not shown). This pattern is seen only under very intense competition and assortment. It appears to be an exaggerated version of the basic sympatric-speciation scenario, with the population resolving itself into a state that tends to minimize competition.

5. DISCUSSION

Mating may often produce a stabilizing component of selection when rare phenotypes are less likely to find com-

patible partners. Thus sympatric speciation by assortative mating is in a bind: strong assortment is needed to cause the population to fission, but it can also generate strong stabilizing selection. Stabilizing selection causes genetic variation to be lost, and it decreases the associations between alleles that are required for one population to split into two. If a population does fission, either in sympatry or in allopatry, then positive frequency-dependent mating success will act against intermediate phenotypes and enhance isolation between the two emerging gene pools. We expect that this qualitative picture also applies to situations in which a mating preference acts on a display trait controlled by a different set of loci.

There is strong empirical evidence for an association between sexual selection and speciation in some groups of animals (Dominey 1984; Barraclough *et al.* 1995; Seehausen 2000). There are several possible explanations for this correlation. Our point is not to question the reality of this pattern, but merely to point out that the sexual selection has many effects, some of which can constrain the possibility of speciation.

Several recent models of Darwin's hypothesis have suggested that sympatric speciation can occur over a much broader range of conditions than we have found. There are three reasons for this discrepancy. The first is the focus of this paper. To avoid the consequences of stabilizing sexual selection, some models explicitly assume that all individuals have the same reproductive success (Kondrashov & Shpak 1998; Dieckmann & Doebeli 1999; Kondrashov & Kondrashov 1999). We suspect that sexual selection may often generate a stabilizing component of selection because some individuals fail to find mates, some individuals mate more often than others, or mating pairs differ in their reproductive success. Second, some models have assumed that the genetic variation among the offspring of a given pair of individuals is fixed, thus implicitly excluding the possibility of fixation (Noest 1997; Van Doorn *et al.* 1998; Drossel & McKane 2000). This assumption is consistent with the classic infinitesimal model of quantitative genetics, which assumes that the trait is under the influence of an infinite number of loci of vanishingly small effect (Bulmer 1980; Bürger 2000). As the number of loci grows, the force of selection on each diminishes, so stabilizing selection does not cause fixation in the case of an infinite number of loci. A third group of studies is based on the hypergeometric model, in which allele frequencies at all loci are constrained to be identical (Doebeli 1996; Kondrashov & Shpak 1998; Kondrashov & Kondrashov 1999; Takimoto *et al.* 2000). This assumption is made to speed numerical simulations, but the equilibria identified can be unstable when allele frequencies at different loci are allowed to vary (Barton & Shpak 2000). It appears that some conclusions about sympatric speciation based on the hypergeometric assumption may be artefacts of this constraint.

Our models treat the strength of mating discrimination as a fixed quantity. This is a reasonable assumption when assortment is a side-effect of mating time, for example, but in other situations the strength of assortment itself might evolve. Beginning with Maynard Smith (1966), several models of sympatric speciation based on this idea have been proposed (reviewed in Kirkpatrick & Ravigné 2002). One can show that modifiers that strengthen positive assortment are generally favoured when selection is disruptive, while disassortative mating is favoured when selection is stabilizing. Given that selection on many traits in nature is stabilizing (Kingsolver *et al.* 2001), the fact that negative assortment is very rare suggests that the evolution of the strength of assortment may also be uncommon. Perhaps assortative mating is most often a by-product rather than an adaptation.

The question of whether mating generates stabilizing selection, however, can be answered only empirically. It is implausible that any real population exactly follows any of the simple mating rules assumed by the models. The most direct way to determine whether or not mating generates a stabilizing component of selection is with studies of phenotypic selection, using for example the methods of Lande & Arnold (1983). In a recent and comprehensive review of studies of selection in nature, Kingsolver *et al.* (2001) found that sexual selection often results in stronger directional selection than does viability selection, but they did not make any generalizations about the strength of the stabilizing (or disruptive) selection it typically produces. It would be valuable to have these data for monogamous as well as polygynous populations, as substantial variation in reproductive success can occur even when every individual mates.

A second major gap in our empirical knowledge is whether the strength of assortative mating in nature is sufficient to drive sympatric speciation. The models suggest that the discrimination between mates needs to be quite strong.

A third empirical issue regards the rules of assortative mating. Our quantitative results depend on the unimodal mating function A given in equation (2.2). Other rules for relative attractiveness are possible. For example, Lande (1981) proposed that some mating preferences might obey an open-ended (or 'psychophysical') rule such that $A(x,y) = \mathrm{Exp}\{mxy\}$. An analysis of the open-ended rule parallel to the one carried out above indicates that it generates very mild *disruptive* selection, which works to promote, rather than inhibit, speciation. It appears likely that this is why Higashi *et al.* (1999), who used this mate-choice rule, found in their simulations that sympatric speciation occurs over a broad range of parameters even though mating was selective. When a single trait, such as phenology, is the basis of assortment, a unimodal mating function such as we used seems more biologically plausible. However, when mating is mediated by a female preference acting on a male display trait (as in Higashi *et al.* 1999), open-ended preferences may be quite common (e.g. Ritchie 1996). We expect the conclusions of this study to apply qualitatively to sympatric speciation based on preference-trait mating systems when mating generates a stabilizing component of selection.

This study has focused on one of the possible scenarios for sympatric speciation, in which disruptive natural selection is caused by competition and reproductive isolation comes from assortative mating. There are other possibilities. Perhaps the most biologically compelling one is based on habitat choice (Bush 1975, 1994; Diehl & Bush 1989; Rice & Salt 1990; Rice & Hostert 1993). Natural selection for divergent habitat preferences can produce strong ecological and genetic isolation at the same time. Habitat choice is, in effect, an extremely efficient form of

assortative mating (Kirkpatrick & Ravigné 2002) and an effective way to maintain polymorphism (Maynard Smith 1966).

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